WHO IS AT RISK

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INTRODUCTION

The prevalence of foot ulceration in the general diabetic population is 4–10%, being lower (1.5–3.5%) in young and highest (5–10%) in older patients. The lifetime risk for foot ulcers in diabetic patients is about 15%. The major adverse outcome of foot ulceration is amputation. Data from several studies have documented that foot ulcers precede approximately 85% of all amputations performed in patients with diabetes. Risk of ulceration and amputation increases 2- to 4-fold with both age and duration of diabetes. According to one report, prevalence of amputations in diabetic patients is 1.6% in the age range 18–44 years, 3.4% among those aged 45–64 years, and 3.6% in patients older than 65 years. Incidence of lower extremity amputations in the United States was 9.8 per 1000 patients with diabetes in 1996, increasing by 26% from 1990, despite efforts to reduce these rates. Data from other countries confirm the increase of amputation rates worldwide. This may be due to aging of the diabetic population, and better reporting. As the diabetic population increases, more amputations are expected in the future.

Foot ulceration and amputation affect the quality of life for patients and create an economic burden for both the patient and the health care system. Therefore, efforts to identify the patient who is at risk for foot ulceration, prevention and appropriate treatment must, of necessity, become a major priority for healthcare providers.

WHICH PATIENTS ARE AT RISK FOR FOOT ULCERATION?

Risk factors for foot ulceration are as follows.

- History of previous foot ulceration or amputation
- Peripheral neuropathy
- Peripheral vascular disease
- Trauma (poor footwear, walking barefoot, objects inside the shoes)
- Foot deformities (prominent metatarsal heads, claw tow, hammer toe, pes cavus, nail deformities, deformities related to previous trauma and surgery, bony prominences, etc.)
- Callus formation
- Neuro-osteoarthropathy
- Limited joint mobility
- Long duration of diabetes
- Poor diabetes control

In addition to these well-recognized risk factors for foot ulceration, several — but not all — studies have shown that foot ulcers are more common in male patients. In addition, social factors including low social status, poor access to healthcare services, poor education and a solitary lifestyle have all been associated with foot ulceration. Another important factor for foot ulceration is poor compliance by the patient with medical instructions and neglecting to follow procedures. Edema may impair blood supply to the foot, particularly in patients with peripheral vascular disease. Inhibition of sweating (anhidrosis) — due to peripheral neuropathy — may cause dry skin and fissures. Dry skin together with limited joint mobility and high plantar pressures contribute to callus formation.

Peripheral neuropathy and vascular disease alone do not cause foot ulceration. It is the combination of the factors mentioned above, that act together in the vast majority of cases. Trauma from either the patient's shoes or from external causes, and loss of protective sensation and peripheral vascular disease are among the major contributors to foot ulceration. Diabetic neuropathy is

the common denominator in almost 90% of diabetic foot ulcers. Trauma initially causes minor injuries, which are not perceived by the patient with loss of protective sensation. As the patient continues his activities, a small injury enlarges and may be complicated by infection. The pathway to foot ulceration in diabetes is depicted in Figure 1.1.

DIABETIC NEUROPATHY

Diabetic neuropathy is defined — according to the International Consensus Group on Neuropathy — as 'the presence of symptoms and/or signs of peripheral nerve dysfunction in people with diabetes, after exclusion of other causes'. The prevalence of peripheral neuropathy in diabetes is 23–42% and is higher (50–60%) among older type 2 diabetic patients. It should be mentioned that the prevalence of symptomatic peripheral neuropathy (burning sensation, pins and needles or allodynia in the feet, shooting, sharp and stabbing pain or muscle cramps at the legs) is only 15–20% and the majority of the patients with neuropathy are free of symptoms. Often, the first sign of peripheral neuropathy is a neuropathic ulcer. Other patients have neuropathic pain and on examination are found to have severe loss of sensation. This combination is described as 'painful-painless legs' and these patients are at increased risk for foot ulceration.

All patients with diabetes should be examined annually for peripheral neuropathy,

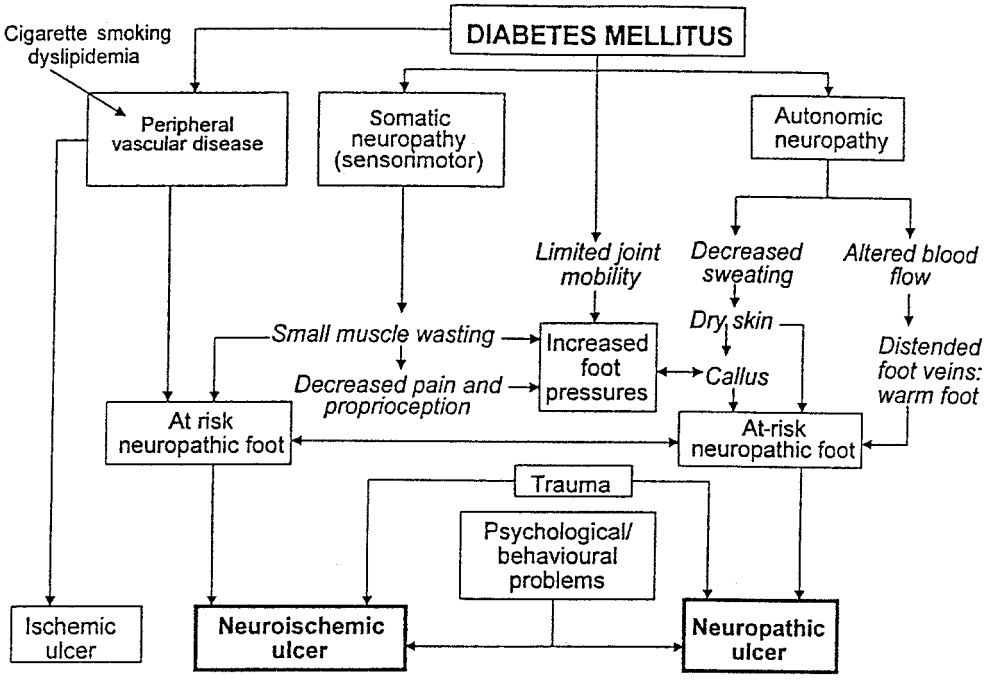

Figure 1.1 Pathways to foot ulceration in diabetic patients. (From Boulton AJM. The pathway to ulceration: Aetiopathogenesis. In Boulton AJM, Connor H, Cavanagh PR (Eds), *The Foot in Diabetes* (3rd edn). Chichester: Wiley, 2000; 61–72, with permission)

so that those at risk for ulceration can be identified. The tests for peripheral neuropathy are many and some of them are quite sophisticated, and are undertaken only in specialist centers. However, the tests that are used to characterize the patient with loss of protective sensation are simple, fast and easily carried out at the outpatient clinic. These tests are as follows.

1. Questioning the patient to ascertain whether symptoms of peripheral neuropathy, as described above, are present. Typically neuropathic symptoms are worse during the night and may wake the patient, who finds relief on walking.
2. Loss of sensation of (a) *pain* (using a disposable pin; this test is carried out only when the skin is intact), (b) *light touch* (using a cotton wisp), and (c) *temperature* (using two metal rods, one at a temperature of 4 °C and the other at 40 °C) on the dorsum of the feet. Typically, in diabetic peripheral neuropathy the sensory deficit is pronounced at the periphery of the extremities (in a 'glove and stocking distribution'). A zone of hypoesthesia is found between the area of loss of sensation and a more central area of normal sensation. Achilles tendon reflexes may be reduced or absent. *Wasting of small muscles* of the feet results in toe deformities (claw, hammer, curly toes) and prominent metatarsal heads. *Vibration perception* is tested using a 128-Hz tuning fork on the dorsal side of the distal phalanx of the great toes (Figure 1.2). A tuning fork should be placed perpendicular to the foot at a constant pressure. During examination the patient is prevented from seeing where the examiner has placed the tuning fork. Examination is repeated twice and there is at least one 'sham' application in which the tuning fork is not vibrating. The patient has normal sensation when his reactions are correct in two out of three tests, but is at risk for ulceration when they are incorrect in two out of the three tests.
3. Pressure perception is tested with Semmes–Weinstein monofilaments. Many studies have shown that inability to perceive pressure is related to a several-fold increase in the risk for foot ulceration. The filaments are available in large sets with varying levels of force required to bend them. Diabetic neuropathy can be detected using the 5.07 monofilament (this filament bends with the application

Figure 1.2 Examination of vibration perception by the use of tuning fork

Figure 1.3 Semmes–Weinstein (5.07) monofilament examination

of a 10-*g* force). Monofilament should be applied perpendicular to the skin surface and with sufficient force so that it bends or buckles (Figure 1.3). Total duration of skin contact of the filament should be approximately 2 s. During examination the patient is prevented from seeing if and where the examiner applies the filament. The patient is asked to say whether he can feel the pressure applied (yes/no) and in which foot (right/left foot). Examination is repeated twice at the same site and there is at least one 'sham' application, in which no filament is applied (a total of three questions per site). The patient has normal protective sensation when the correct answer is given for two out of the three tests and is at risk for ulceration when they are not. The International Consensus on the Diabetic Foot suggested three sites to be tested on both feet: the plantar aspect of the great toe, the first and the fifth metatarsal heads. The filament must be applied at the perimeter and not at an ulcer site, callus, scar or site of necrotic tissue.

4. Determination of vibration perception thresholds using a biothesiometer or a neurothesiometer. Vibration perception threshold is measured at the tip of the great toes with the vibrating head of the device balanced under its own weight (Figure 1.4). The vibrating stimulus is increased until the patient feels it, the stimulus is then withdrawn and the test repeated. This test is usually carried out three times at each site and the mean value is calculated. Several studies have shown that a vibration perception threshold over 25 V is associated with a 4- to 7-fold increase in risk for foot ulceration.

PERIPHERAL VASCULAR DISEASE

ASSESSMENT OF THE VASCULAR STATUS IN PATIENTS WITH DIABETES

The prevalence of peripheral vascular disease in diabetic patients is 15–30%. The

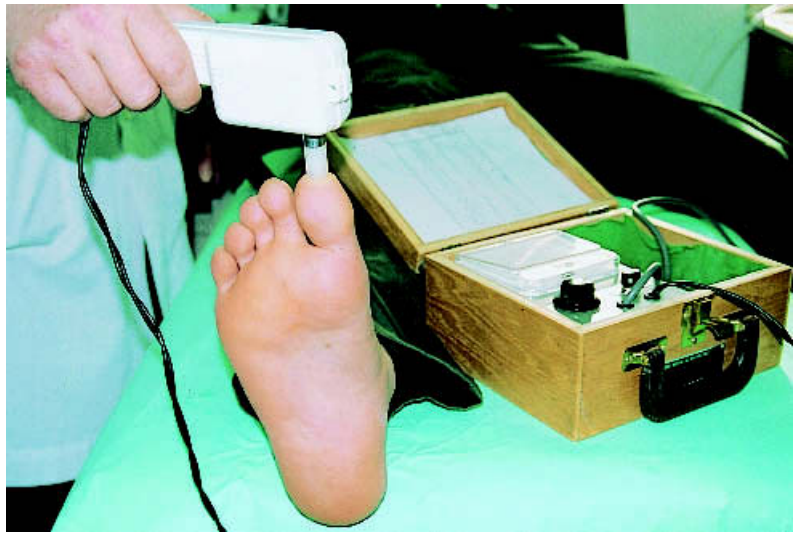

Figure 1.4 Examination of vibration perception by the use of a biothesiometer

disease progresses with both duration of diabetes and age. A diagnostic work-up of the peripheral extremities is based on clinical examination (history of intermittent claudication, rest pain, walking distance, palpation of leg pulses, and measurement of ankle brachial index). Co-existence of neuropathy in diabetic patients might reduce the pain of intermittent claudication or even ischemic rest pain. Palpation of feet pulses remains the cornerstone of screening for peripheral vascular disease. The absence of two or more pulses on both feet is diagnostic of peripheral vascular disease. Based on the results of clinical examination, a decision must be made as to whether the doctor will proceed with more sophisticated methods of examination of the lower extremities in order to determine the exact level and degree of the arterial obstruction.

Fontaine Clinical Staging

Fontaine clinical staging of peripheral arterial disease includes four stages:

Stage I is asymptomatic; patients may complain of numbness or that their legs get easily tired, but they do not seek medical help. Usually the superficial femoral artery is stenosed at the level of the Hunterian duct; lateral circulation of the deep femoral artery is adequate for the needs of the limb.

Stage II in which patients suffer from intermittent claudication; they are subclassified as *Stage IIa*, if they can walk without symptoms for more than 250 m; or *Stage IIb*, if they have to stop earlier. If patients feel pain in the leg, it is usually due to occlusion of the femoral artery, while an occlusion of the iliac artery causes pain in the thigh.

Stage III patients suffer from rest pain of the limb, which may become constant and very intense, usually during the night; the pain is often resistant to analgesics. The prognosis is not good; half of these patients will have an amputation within the next 5 years.

Stage IV patients have gangrene. Minor trauma, ulcers or paronychias may evolve

into gangrene when stage III peripheral artery disease is present. The patient feels pain at rest unless diabetic neuropathy is also present.

Noninvasive Vascular Testing

Calculation of the Ankle Brachial Index (ABI). The ankle brachial index (ankle arm index) is widely used and can easily be measured in the outpatient clinic. Measurements are made with the use of a pocket-size continuous-wave Doppler probe operating at 4 or 10 MHz. The brachial systolic pressure on both sides is determined first. Then the ankle systolic pressure on each side is determined with the Doppler probe by applying a blood pressure cuff around the ankle, just above the malleolus. Ankle pressure is measured at both posterior tibial (behind the medial malleolus) and dorsal pedal arteries. No pressure is applied on the probe. Pressures are determined at a beam-vessel angle of approximately 60°. After measuring the systolic pressures, the highest ankle pressure is divided by the highest brachial pressure; this ratio is called the *ankle brachial index* (ABI). Occasionally, no audible signal can be obtained from the foot arteries. In these cases, a careful search often reveals a peroneal collateral signal anteriorly, next to the lateral malleolus. Normally, systolic ankle pressure exceeds systolic arm pressure by 12–24 mmHg. The normal value of the ABI is 1 to 1.2. A level of less than 0.9 is usually taken as indicative of occlusive arterial disease. An ankle systolic pressure of <50 mmHg or an ABI <0.3 in the presence of rest pain or tissue damage denotes critical limb ischemia. The equivalent toe systolic pressure is 30 mmHg or less.

A change of >0.15 in the ABI during follow-up suggests significant narrowing and it is an indication for further study with angiography. A spontaneous rise in the ABI is usually attributable to the development of collateral circulation.

Medial calcification, which is very common in diabetes (Figure 1.5), renders the underlying arteries incompressible, resulting in spuriously high ABI values (more than 1.2). In these cases, the severity of arterial occlusive disease can be assessed by toe pressure measurements. Other causes of inaccurately high ABI values include too high positioning of the upper body, chronic venous insufficiency and significant ankle edema. Spurious low ABI values can result from the rapid deflation of the cuff, excessive probe pressure, and an insufficient rest period.

Despite these limitations, the ankle brachial index is a useful screening tool for the assessment of presence and severity of peripheral vascular disease and it remains the basic examination suggested by an international panel on the assessment of peripheral vascular disease in diabetes (see below).

Toe Pressures. Toe pressures are measured by a pneumatic cuff with a diameter which

Figure 1.5 Extensive calcification of the posterior tibial artery

is about 1.2 times that of the digit, wrapped around the proximal phalanx, with a flow sensor (usually a photoplethysmograph) located distally (Figure 1.6). In addition, toe pressures can also be measured using a digital strain gauge. Normal toe pressures average 24–40 mmHg or less compared to ankle pressure. Rest pain, skin lesions, or both are present in approximately 50% of limbs with toe pressures $\leq$30 mmHg, and in a much lower proportion of patients with toe pressures above this level. Toe pressures do not differ between patients with and without diabetes. Spuriously high toe pressures due to arterial calcification seldom occur at the toe level. For this reason, toe pressure determination is valuable in diabetic patients when an ankle pressure is abnormally high.

Transcutaneous Oximetry. Transcutaneous oximetry (measurement of transcutaneous oxygen pressure, $TcPO_2$) is used for the assessment of severe peripheral vascular disease. It is usually measured at the dorsum of the feet with the patient in the supine position (Figure 1.7). With increasing age, the $TcPO_2$ tends to decrease,

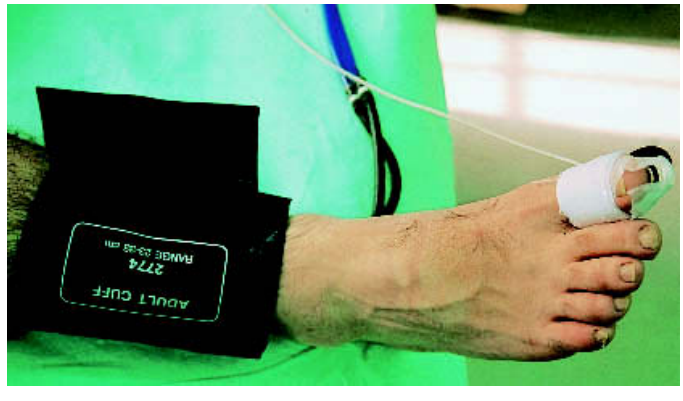

Figure 1.6 Toe and ankle pressure measurement

Figure 1.7 Transcutaneous oximetry

paralleling a similar decline in arterial PO_2. Normal subjects have values of 40 to 70 mmHg. In general, a resting $TcPO_2$ greater than 55 mmHg may be considered normal, regardless of age. Patients with anemia may also have lower values. Patients with rest pain or gangrene have values between 0 and 30 mmHg. In diabetes, $TcPO_2$ is lower than in age-matched arteriopathic patients. A $TcPO_2$ <40 mmHg is associated with failure of wound healing, while an increase after angioplasty or bypass surgery predicts success of the intervention. Because the results are not affected by arterial calcification, this method is particularly valuable for evaluating diabetic vascular disease.

Segmental Pressures Measurement. Abnormal blood pressure values found by any of the above methods indicate that arterial occlusive disease is present, but they do not identify the specific segments involved. Further diagnostic information can be obtained by measuring pressure gradients in the legs. However, only rarely do these measurements need to be made when the ABI is normal. To determine segmental pressure in the legs, pressure cuffs 10–12 cm wide are applied around the thigh at the groin level, above the knee, below the knee and at the ankle level (Figure 1.8). By listening with a Doppler probe over the pedal arteries (posterior tibial or dorsal pedal), the pressure at the level of the inflated cuff can be measured. A pressure index can be obtained by dividing the segmental systolic pressure by the brachial pressure. The pressure index should be equal to 1.0 or slightly higher. Normal pressure index at the high thigh level is 1.3. The pressure gradient between any two adjacent levels in the normal leg is <20–30 mmHg. Gradients >30 mmHg suggest that a significant stenosis is present at the intervening arterial segment. When the gradient exceeds 40 mmHg the artery is occluded.

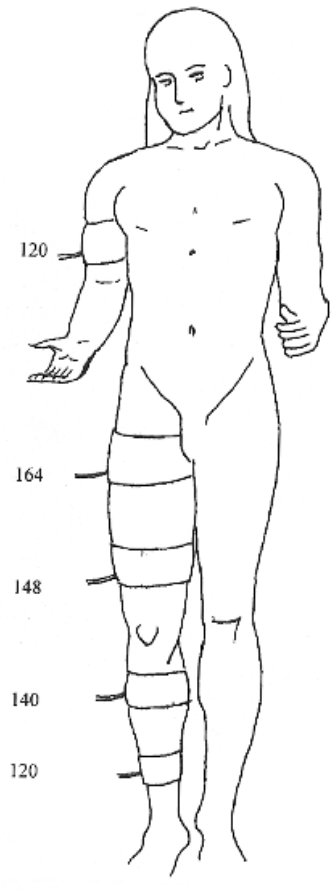

Figure 1.8 Normal segmental pressures. The pressure gradient between any two adjacent levels in the normal leg is less than 20–30 mmHg. Gradients greater than 30 mmHg suggest that a significant stenosis is present at the intervening arterial segment. When the gradient exceeds 40 mmHg the artery is occluded

It should be taken into account that patients with severe stenosis at a proximal level (e.g. aortoiliac disease) may have spuriously normal pressure gradients between high thigh and low thigh in the presence of severe superficial femoral artery stenosis.

In addition, obstructions below the knee may not be diagnosed, unless the stenosis is sufficiently severe to involve all three tibial arteries.

Segmental Plethysmography. Plethysmography is a useful technique for the assessment of peripheral arteries. There are several types of plethysmographs (air, mercury, indium–gallium and strain gauge plethysmographs) and all measure the same parameter: the momentary change in the volume of the soft tissues when a pulse wave fills the arteries of the area of the leg which is being examined. Photoplethysmography measures blood concentration in the cutaneous microcirculation by detecting the reflection of the applied infrared light. Air plethysmographs are the standard instruments for segmental plethysmography. Pressure cuffs are applied at different levels of the leg as in segmental pressure measurement. A plethysmograph records the change in volume as a wave, which reflects the intra-arterial changes. The normal segmental volume pulse contour is characterized by a steep, almost vertical upstroke, a sharp systolic peak, and a down slope that bows towards baseline during diastole. In the middle of the down slope there is a prominent dicrotic wave. Distal to a stenosis, the upslope is less steep, the peak becomes rounded, the down slope bows away from the baseline, and the dicrotic wave disappears. Examples of various degree of arterial stenosis are shown in Figure 1.9. A plethysmography record is not affected by the presence of arterial calcification; for this reason it is a valuable method for the assessment of peripheral vascular disease in diabetes.

Ultrasonography. Arterial ultrasound examination has become very popular in recent years. It is a simple, low cost and valid method for determination of the site and degree of obstructive lesions, and of the patency of a vessel after revascularization. The site of an arterial stenosis can be identified by serial placements of the Doppler probe along the extremities. However, there is no justification for its use as a routine screening procedure. The exact site of arterial disease is located by the

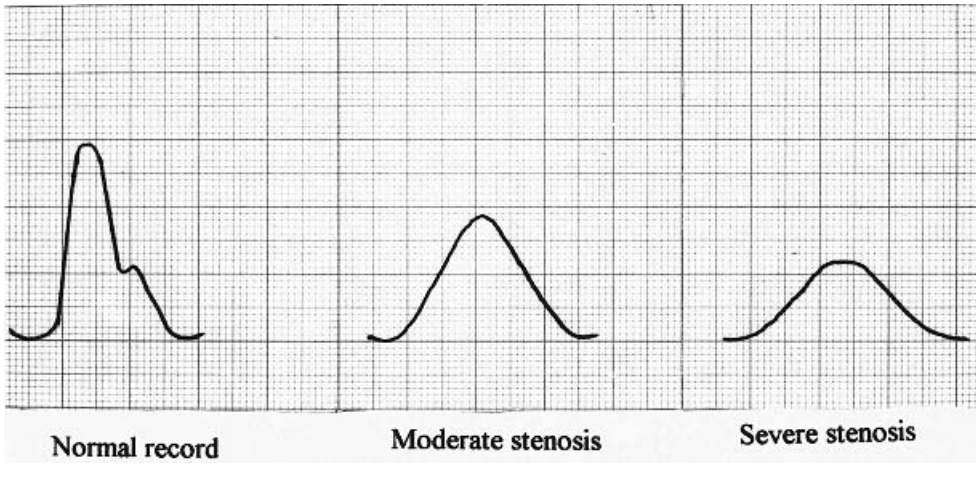

Figure 1.9 Plethysmography pulse volume waveforms associated with different degrees of peripheral vascular disease

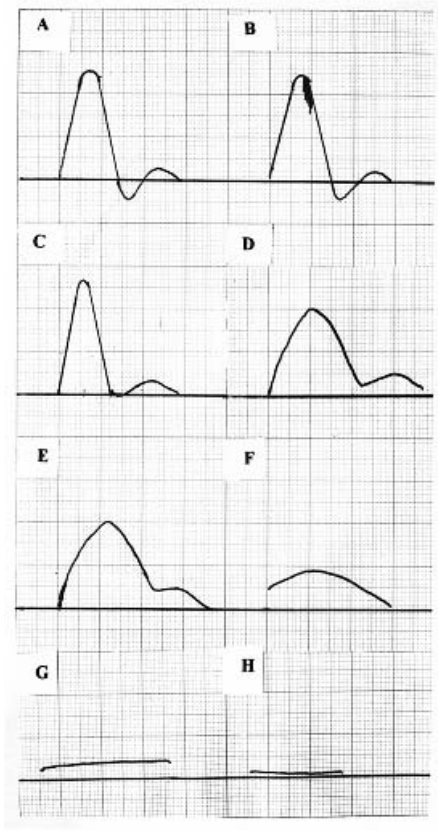

Figure 1.10 Qualitative analysis of spectral waveforms proximal to the site of the probe. (**A**) Normal. (**B**) Mild arterial stenosis causing turbulence during systole. (**C** and **D**) Loss of reverse flow due to more severe stenosis. (**E** and **F**) As the degree of stenosis increases, the rate of acceleration of the upstroke decreases, the peak becomes rounded (**E**) and the wave becomes continuous and less pulsatile (**F–H**). Completely damped waveforms (**F–H**) in the pedal arteries are compatible with multilevel vessel disease and indicate the presence of blood flow due to the development of collateral circulation

use of duplex scanning. Duplex scanners use the combination of real time B-mode ultrasound imaging of the arterial wall, together with pulsed Doppler imaging and examine flow patterns in a defined area within the artery lumen. Pulsed Doppler imaging produces spectral analysis of the pulse wave which delineates the complete spectrum of frequencies (that is, blood flow velocities) found in the arterial waveform during a single cardiac cycle. Tissues are displayed in varying shades of gray (duplex) on the screen. The addition of color frequency mapping (color duplex or triplex) makes identification of arterial stenosis easier and allows a better description of the atheromatous plaques on the arterial wall. A normal spectrum shows a typical triphasic flow pattern, consisting of a steep systolic upstroke, a systolic peak, a reverse flow component in early diastole and a pre-systolic zero flow (Figure 1.10). A clear spectral window under the systolic peak is a normal finding, signaling the absence of slow turbulent flow components. If a stenosis is present the window becomes occluded. The degree of stenosis can be quantified by analyzing the spectral waveform, and by determining the peak systolic velocity ratio (PSV ratio). In general, a cross-sectional reduction of at least 30% must be present to produce a detectable spectral change. The flow velocities may vary, but peak systolic velocities in the arteries above knee are about 50–100 cm/s, while below knee they are approximately 50 cm/s.

Qualitative analysis of the waveform. Inspecting the contour of the spectral waveform is of considerable diagnostic value.

Table 1.1 Peak systolic velocity ratio (PSV ratio) for the determination of the degree of stenosis

PSV ratio	Reduction in cross-sectional area
<2.5	0–49%
>2.5	50–74%
>5.5	75–99%

Atherosclerotic disease proximal to the site of the probe produces a subtle change in the contour of the systolic peak or in the early deceleration phase (Figure 1.10). With increasing proximal stenosis, the reverse flow component is damped and then disappears entirely.

Quantitative analysis of the waveform. The most widely used criterion for

Table 1.2 Criteria for lower limb arterial stenosis in spectral analysis

Percentage stenosis	Pre-stenotic spectrum	Intra-stenotic spectrum	Spectrum just past the stenosis
0–50%	Normal: — Triphasic or biphasic — Narrow frequency band — Clear spectral window	Increase in PSV (by <100% and/or <180 cm/s)	No significant turbulence Possible flow reversal
51–75%	Normal	Increase in PSV (by >100% and/or >180 cm/s)	Flow reversal Possible slight turbulence
76–99%	Normal or slightly reduced velocity	Increase in PSV (by >250% and/or >180 cm/s)	Significant turbulence Complete occlusion of spectral window

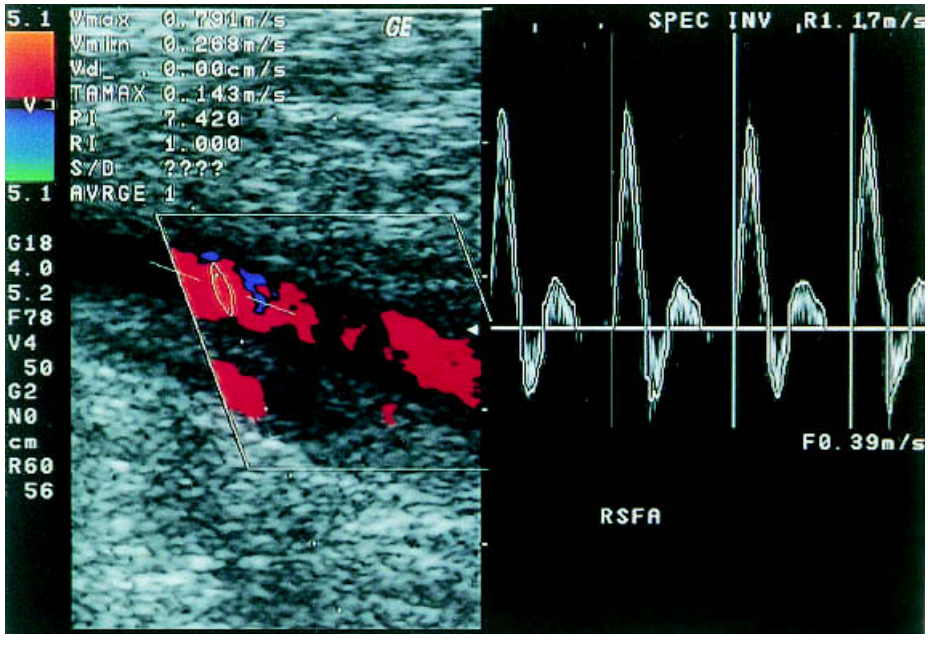

Figure 1.11 Normal triphasic spectral waveform from the right superficial femoral artery. Note the narrow, steep increase and decrease of the waveform. Peak systolic velocity is 79.1 cm/s (normal peak systolic velocities in the arteries above knee are 50–100 cm/s). (Courtesy of C. Revenas)

diagnosis of peripheral arterial stenosis is the peak systolic velocity ratio. This ratio expresses the relationship of the intrastenotic peak systolic velocity to the lowest post-stenotic or pre-stenotic peak systolic velocity. The PSV ratio allows estimation of the degree of a stenosis without distortion by a second stenosis located at a more distal or a proximal site (Table 1.1). Other criteria used for the estimation of arterial stenosis are presented in Table 1.2.

Duplex ultrasonography has a sensitivity of 80% and specificity above 90% for detecting femoral and popliteal stenosis compared with angiography, but it is less reliable for the assessment of the severity of stenosis in the tibial and peroneal arteries. Normal and abnormal spectral waveform recordings are shown in Figures 1.11–1.18.

Other Methods. Modern methods for the assessment of peripheral arteries include helical or spiral computed tomography (CT) and magnetic resonance angiography. Spiral CT has the ability to generate three-dimensional images and is most useful in the evaluation of large arteries (e.g. thoracic or abdominal aorta). Disadvantages include intravascular administration of iodinated contrast material and the inability to assess small vessel disease. Magnetic resonance angiography (MRA) is mainly used for examining the cerebral vessels and the carotid arteries. Recent data suggest that this method might replace angiography as a primary imaging examination for

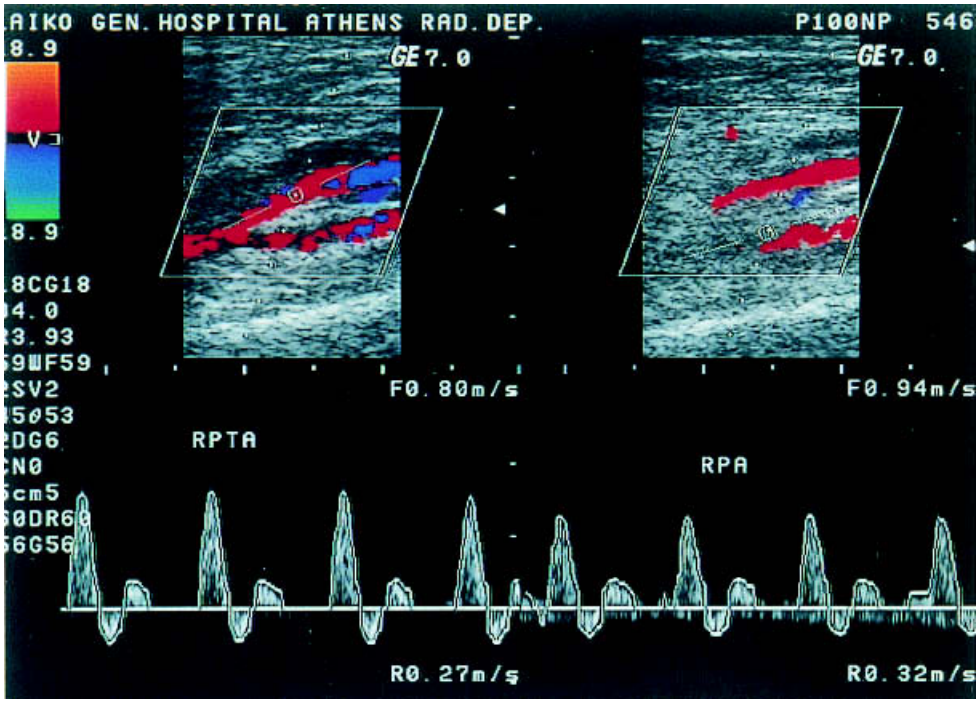

Figure 1.12 Normal triphasic spectral waveform from the right posterior tibial artery. At the top of the figure the duplex scan of the artery is seen. Peak systolic velocity is 49 cm/s. (Courtesy of C. Revenas)

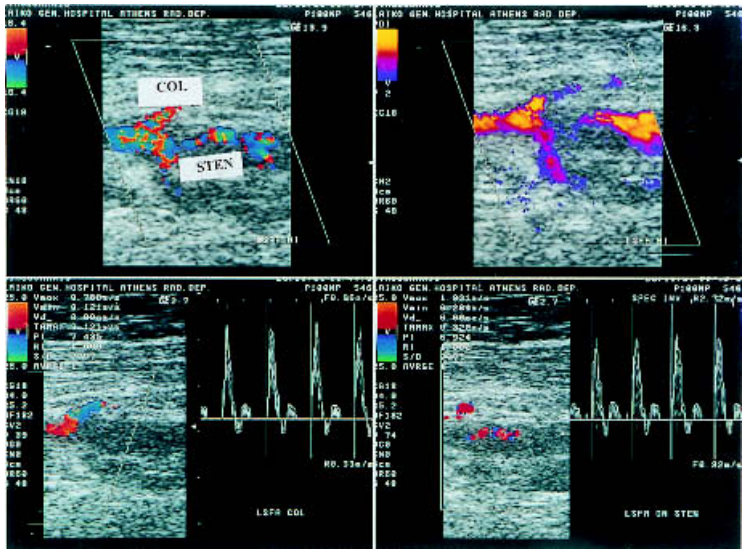

Figure 1.13 In the left upper panel a significant stenosis (STEN) of the left superficial femoral artery with collateral circulation development (COL) is shown. Note (left lower panel) the triphasic spectral waveform in the collateral vessel and that the peak systolic velocity is 78 cm/s, which is too high for such a vessel. In the area of the femoral artery stenosis, the peak systolic velocity is high (193 cm/s) and the waveform is triphasic, but blood flow during diastole is low, as seen from the short duration of the reverse flow (right lower panel). Adjacent to the spectral waveform, a color duplex scan of the artery with the stenosis is shown. These findings suggest the presence of stenosis of approximately 50–80%. In the upper right panel a dynamic Doppler recording is shown, which gives a clearer image of the collateral vessels. (Courtesy of C. Revenas)

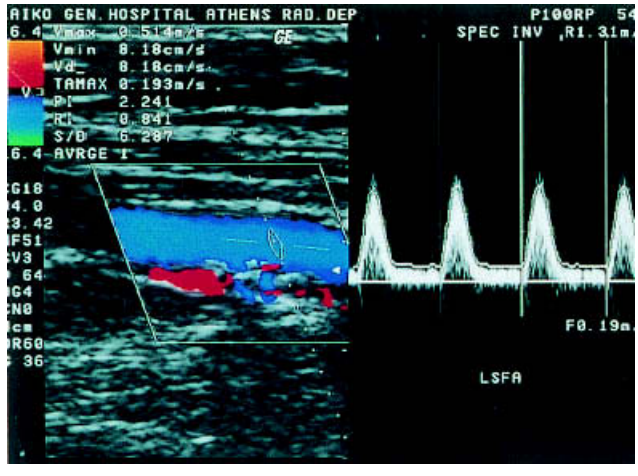

Figure 1.14 Biphasic spectral waveforms obtained from the left superficial femoral artery. The spectral window is widened and is filled in, although not completely. Peak systolic velocity is low (51.4 cm/s). These findings indicate the presence of significant proximal stenosis at one or multiple levels. (Courtesy of C. Revenas)

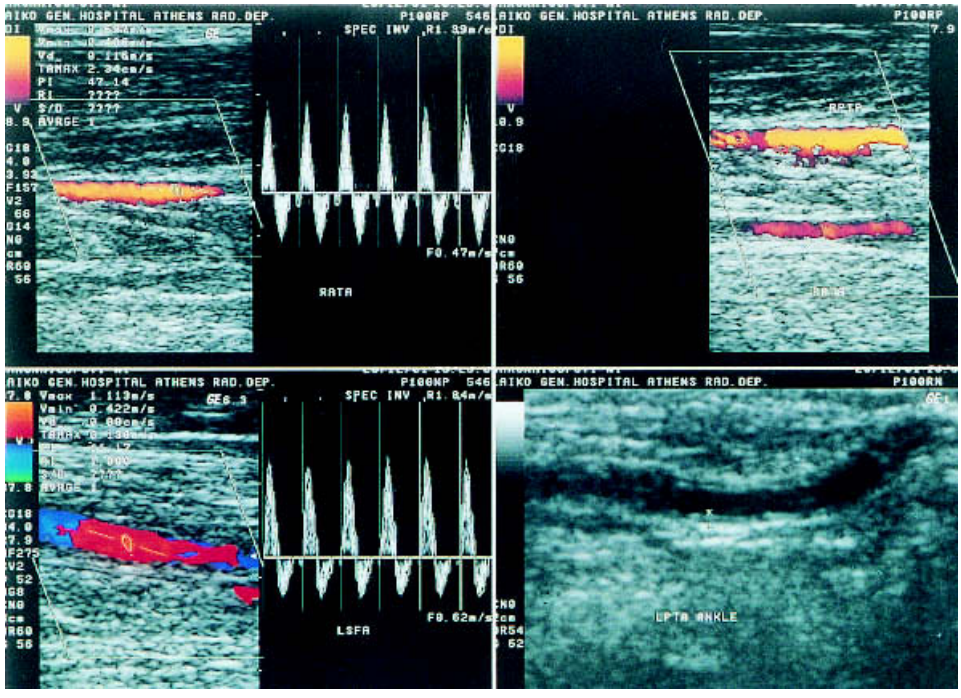

Figure 1.15 The spectral waveform from the right anterior tibial artery in an area of stenosis is seen in the upper left panel. Peak systolic velocity is high (69.7 cm/s) — peak systolic velocities in the arteries below knee are normally ~50 cm/s — and there is mild widening of the spectrum during both systole and diastole. This record corresponds to a stenosis of about 30%. The spectral waveform from the left superficial femoral artery is shown in the lower left panel. There is mild spectral widening, and loss of pre-systolic flow. The color duplex image of the right tibial arteries is shown in the right upper panel. A duplex scan of the left posterior tibial artery is shown in the lower right panel. (Courtesy of C. Revenas)

peripheral vascular disease. Angiography may be reserved for percutaneous interventions and in cases of equivocal findings only. In addition, an MRA is a simple, nontoxic and relatively inexpensive method.

An International Meeting on the Assessment of Peripheral Vascular Disease in Diabetes was held in 1993 and made the following recommendations for the detection and follow-up of peripheral vascular disease.

- All adults (age $\geq$ 18 years) with diabetes should be asked whether they suffer from intermittent claudication. Presence of claudication is an indication for ankle brachial index (ABI) determination on an annual basis. If the ABI is less than 0.9, the patient needs intensive management of cardiovascular risk factors. All patients with lifestyle-limiting claudication should be referred for specialist vascular assessment. Intensive management of cardiovascular risk factors includes reduction of lipid levels, smoking cessation, control of blood pressure, weight and glucose levels and the use of aspirin as in coronary heart disease.
- All adults (age $\geq$ 18 years) with diabetes should be examined annually for signs of

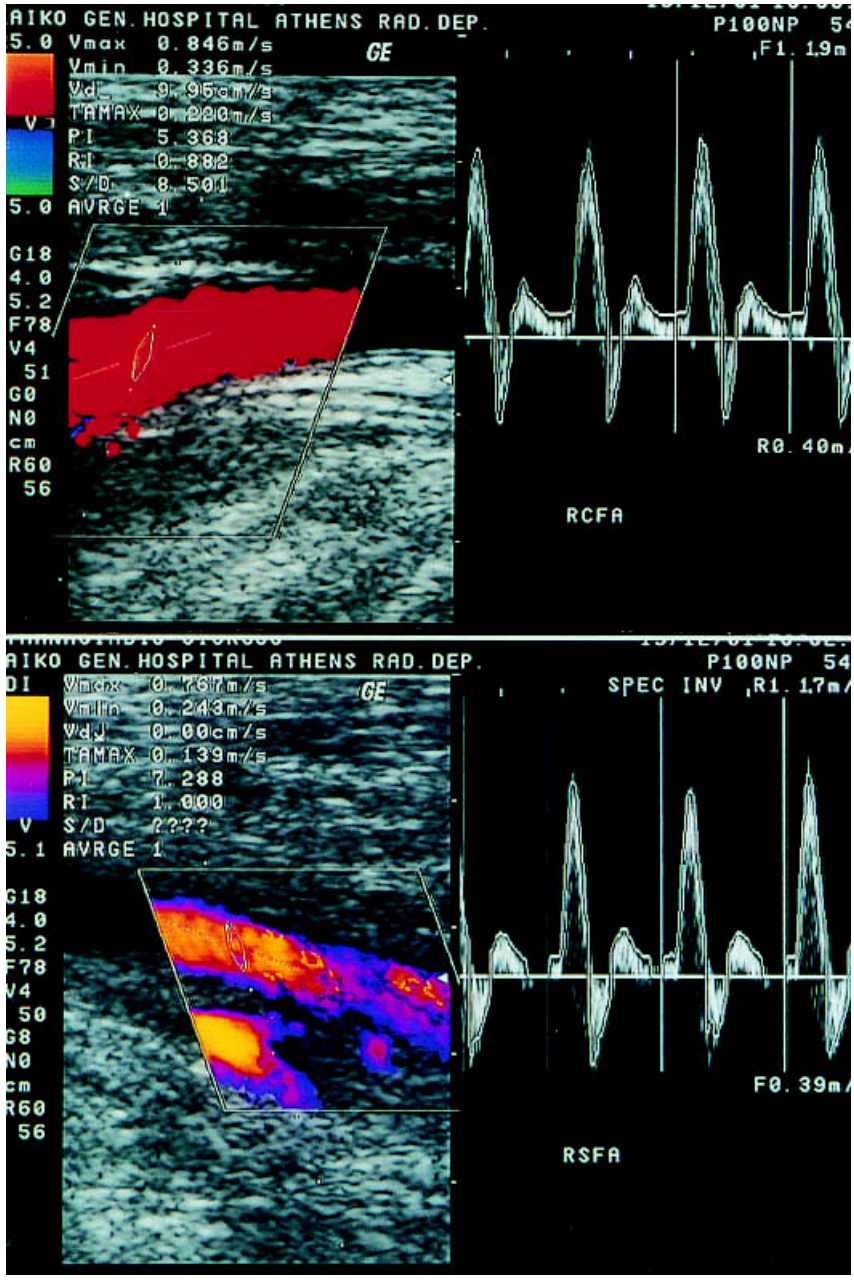

Figure 1.16 Near normal spectral waveforms obtained from the right common (upper panel) and right superficial (lower panel) femoral arteries. The peak systolic velocity is reduced slightly; the waveform is triphasic and there is minimal widening of the spectral window. These findings suggest the presence of a mild proximal stenosis. (Courtesy of C. Revenas)

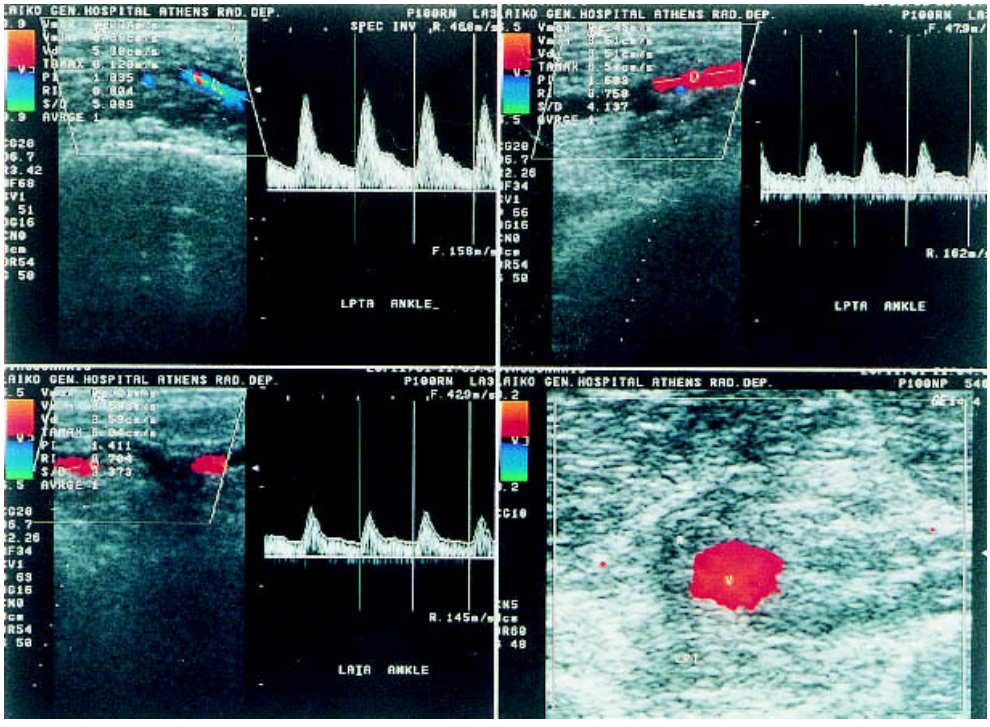

Figure 1.17 Upper left panel: a biphasic waveform of the left posterior tibial artery at ankle level. The peak systolic velocity is reduced (27.4 cm/s) and there is widening of the spectral window during systole, while velocity is high during diastole. The artery diameter is normal as seen in a color duplex image on the left of the spectral waveform. These findings suggest the presence of a proximal stenosis of about 40%. Right upper panel: the same artery at another site after a stenosis. The low peak systolic velocity (14.5 cm/s), biphasic waveform, and spectral widening during systole, as well as the high velocity during diastole are notable features. These findings suggest the presence of a proximal stenosis of more than 50%. Left lower panel: duplex scan of the left anterior tibial artery from the same patient and the recorded spectral waveform. An even lower peak systolic velocity (12.1 cm/s), significant widening of the systolic spectral window and high diastolic velocity are shown. The diameter of the artery is normal (lower right panel). The above findings signify the presence of a proximal stenosis of about 50–60%. (Courtesy of C. Revenas)

critical limb ischemia (gangrene, ulcer, skin changes, or ischemic rest pain). If such signs are present, the patient should be referred for specialist vascular assessment. In addition, intensive management of co-existent cardiovascular risk factors should be initiated.

- Palpation of the dorsalis pedis and posterior tibial artery as well as auscultation for femoral artery bruits should be performed on an annual basis for all adults with diabetes. If one pedal artery is absent or diminished or if bruits are audible, ABI determinations should be carried out annually. If the ABI value is below 0.9, intensive management of co-existent cardiovascular risk factors should be initiated.
- Patients for whom ABI monitoring is recommended: (a) all those with type 1

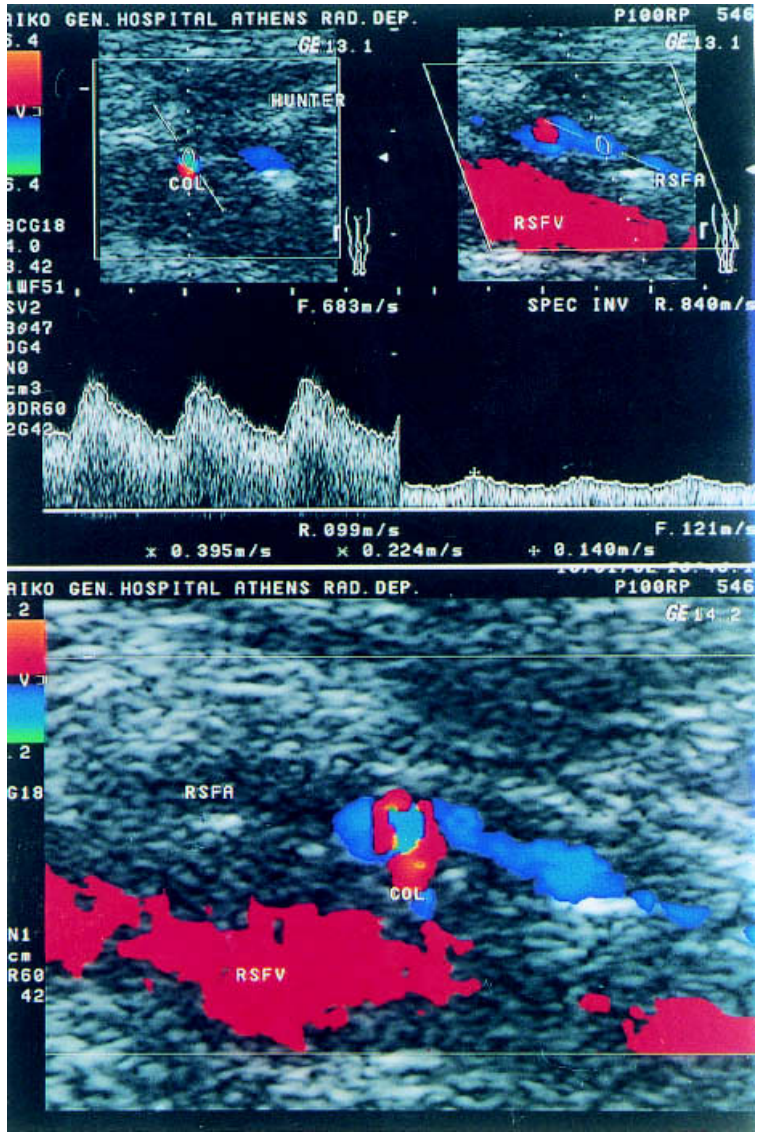

Figure 1.18 Lower panel: complete obstruction of the right superficial femoral artery (RSFA) at the canal of Hunter. A collateral vessel (COL) is seen proximal to the stenosis. Distal to the site of the obstruction there is blood flow in the superficial femoral artery from collateral vessels. Upper panel: the spectral waveform obtained from the collateral vessel shown in the lower panel of the figure. The waveform is biphasic, both peak systolic and diastolic velocities are high and there is widening of the systolic spectral window. The waveform obtained from the right superficial femoral artery distal to the site of the complete obstruction is shown. Notice the low peak systolic and the high diastolic velocity. This waveform is called *tardus pardus.* This type of spectral waveform is similar to that obtained from the venous circulation, and signifies blood flow in an artery resulting from the development of collateral circulation. As more collateral vessels fill the artery, the spectral waveform may be triphasic, but the peak systolic velocity will be reduced. (Courtesy of C. Revenas)

diabetes older than 35 years, or who have had diabetes for over 20 years at baseline; (b) all patients older than 40 years at baseline with type 2 diabetes; (c) any diabetic patient who has newly detected diminished pulses, femoral bruits, or a foot ulcer; (d) any diabetic patient with leg pain of unknown etiology.

- Based on the results of the ABI, the following recommendations are suggested:
  - ➢ If the ABI is above 0.9, measurement should be repeated every 2–3 years.
  - ➢ If the ABI is 0.50–0.89, measurement should be repeated within 3 months and intensive management of co-existent cardiovascular risk factors should be initiated.
  - ➢ If the ABI is below 0.5, the patient should be referred for specialist vascular assessment and intensive management of co-existent cardiovascular risk factors should be initiated.
- If an incompressible artery with an ankle pressure above 300 mmHg or an ankle pressure 75 mmHg above arm pressure is found, these measurements should be repeated in 3 months. If still present, these patients should be referred for vascular assessment and intensive management of co-existent cardiovascular risk factors should be undertaken.

Invasive Vascular Testing — Arteriography

Arteriography remains the definitive diagnostic procedure before any form of surgical intervention. It should not be used as a diagnostic procedure to establish the presence of arterial disease. Contrast material may exaggerate any preexisting renal disease and for this reason the contrast material used should be limited as much as possible. In addition, the International Meeting on the Assessment of Peripheral Vascular Disease in Diabetes strongly recommended that in diabetic patients arteriography should be carried out before any decision regarding an amputation is made, in order to assess the exact status of the vascular tree, particularly when the ankle brachial index and toe systolic pressure indicate that arterial disease is present.

Keywords: Etiopathogenesis of foot ulceration; diabetic neuropathy, diagnosis; symptoms of peripheral neuropathy; vibration perception threshold; Semmes–Weinstein monofilaments; assessment of vascular status; ankle brachial index; medial arterial calcification; toe pressure; transcutaneous oximetry; segmental pressures measurement; segmental plethysmography; ultrasonography; duplex; triplex; waveforms, quantitative analysis; waveforms, qualitative analysis; peak systolic velocity ratio; spiral computed tomography; magnetic resonance angiography; invasive vascular testing; angiography; Fontaine stage

BIBLIOGRAPHY

1. The International Working Group on the Diabetic Foot. *International Consensus on the Diabetic Foot*. Amsterdam, The Netherlands, 1999.
2. Boulton AJM, Greis FA, Jervell JA. Guidelines for the diagnosis and outpatient management of diabetic peripheral neuropathy. *Diabet Med* 1998; **15**: 508–514.
3. Boulton AJM. The pathway to ulceration: Aetiopathogenesis. In Boulton AJM, Connor H, Cavanagh PR (Eds), *The Foot in Diabetes* (3rd edn). Chichester: Wiley, 2000; 61–72.
4. Veves A, Uccioli L, Manes C, Van Acker K, Komninou H, Philippides P, Katsilambros N, De Leeuw I, Menzinger G, Boulton AJ. Comparison of risk factors for foot problems in diabetic patients attending teaching outpatient clinics in four different

European states. *Diabet Med* 1994; **11**: 709–713.
5. Ziegler RE, Summer DS. Physiologic assessment of peripheral arterial occlusive disease. In Rutherford RB (Ed.), *Vascular Surgery* (5th edn). Philadelphia: Saunders, 2000; 140–165.
6. Katsilambros N, Hatzakis A, Perdikaris G, Pefanis A, Papazachos G, Papadoyannis D, Balas P. Peripheral occlusive arterial disease in longstanding diabetes mellitus. A population study. *Int Angiol* 1989; **8**: 36–40.
7. Kasilambros NL, Tsapogas PC, Arvanitis MP, Tritos NA, Alexiou ZP, Rigas KL. Risk factors for lower extremity arterial disease in non-insulin-dependent diabetic persons. *Diabet Med* 1996; **13**: 243–246.
8. Donnelly R, Hinwood D, London NJM. Non-invasive methods of arterial and venous assessment. In Donnelly R, London NJM (Eds), *ABC of Arterial and Venous Disease*. London: BMJ Books, 2000; 1–4.
9. Orchard TJ, Strandness DE. Assessment of peripheral vascular disease in diabetes. *Diabetes Care* 1993; **16**: 1199–1209.
10. Veves A, Giurini JM, LoGerfo FW. *The Diabetic Foot: Medical and Surgical Management*. Totowa, NJ: Humana Press, 2002.